

## Back to Basics:

# Mechanism of Action of the Vacuum Assisted Closure Device

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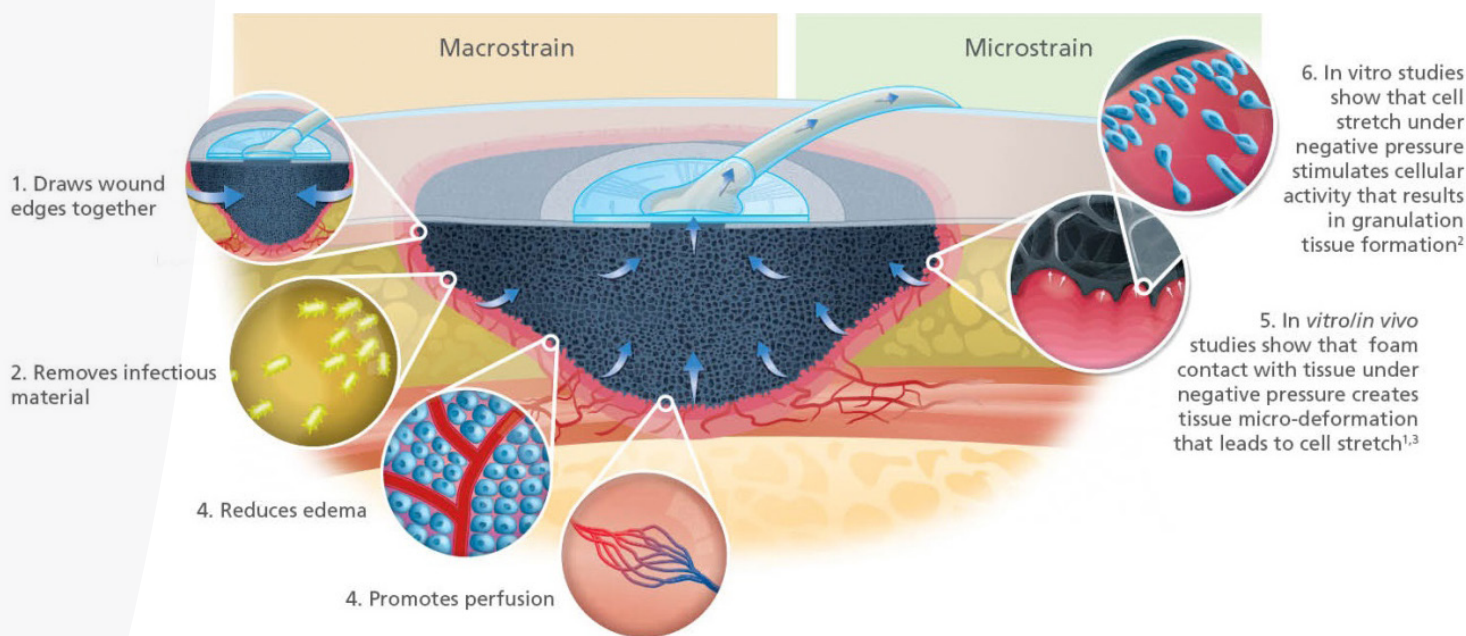
INTRODUCED by KCI in 1995, Vacuum Assisted Closure® (V.A.C.®) devices have had an impact on the healing of wounds. Drs. Louis Argenta and Michael Morykwas from Wake Forest University had worked for years on several prototype devices where suction was used to facilitate wound closure. This included devices that used a CPR mask to close the wound and connect it to suction. One of the devices they found most promising was when they used a highly porous polyurethane foam beneath a semi-occlusive dressing and connected this to suction. My colleague, Karl Breuing, MD, introduced this to our hospital (Brigham

and Women's Hospital) at that time and had some amazing results. Soon after, the other plastic surgeons in our hospital, followed by surgeons of other disciplines, adopted this remarkable technology.

We first noticed a robust granulation tissue that formed when the device was placed onto wounds. The appearance of this seemed to mimic the porosity of the surface of the polyurethane foam. We wondered how this worked. As we asked colleagues, most people felt that it was because we were removing fluid from the wound and taking the "bad stuff" out. We noticed this same response in several other wounds where there was minimal

fluid drainage and wondered if there was another mechanism involved

Quentin Eichbaum, MD, PhD, was a third-year medical student at Harvard Medical School and saw a patient (on whom he needed to do a report) with a V.A.C.® Therapy device. He put together a team from Harvard Medical School, including Donald Ingber, MD, PhD, and some MIT graduate students, to propose additional mechanisms to explain what we were seeing in the clinic. We constructed a finite element model of the wound and its interaction with the foam. The model predicted that the foam would deform the wound, and, according to previous work



1. Saxena SM, et al. Vacuum Assisted Closure: Microdeformations of Wounds and Cell Proliferation. *Plastic & Reconstructive Surgery*, 2004;114(5):1086-1095
2. McNulty AK, et al. Effects of negative pressure wound therapy on the fibroblast viability, chemotactic signaling and proliferation in a provisional wound (fibrin) matrix. *Wound*, 2007; 15:838-846
3. McNulty AK, et al. Effects of negative pressure wound therapy on cellular energetics in fibroblasts grown in a provisional wound (fibrin) matrix. *Wound Repair and Regeneration*. 2009 Mar;17(3):192-9.

of Dr. Ingber, this deformation would also deform cells, prompting them to divide and proliferate. This theory we refer to as microdeformation.<sup>1</sup>

Many had also noted that these devices draw the wound edges together; this is referred to as macrodeformation. Others have proposed that keeping a wound sealed allows it to be warm and moist, which is well known to contribute to wound healing.

Undoubtedly, there are several mechanisms at work in the application of V.A.C.<sup>®</sup> Therapy devices. We and many others are in the process of unraveling the mechanism of these devices with the hope that this improved understanding will lead to better application of the existing devices and provide clues as how to better design improved devices in the future.

In summary, we believe that six primary mechanisms of V.A.C.<sup>®</sup> Therapy

devices are: 1) macrodeformation; 2) microdeformation; 3) removal of fluids; 4) reduction of edema, 5) promotion of wound bed perfusion, and 6) keeping the wound covered so it maintains constant moisture content. We look forward to learning much more about these mechanisms in the future.

References:

1. Saxena V, Hwang CW, Huang S, Eichbaum Q, Ingber D, Orgill DP. Vacuum-Assisted Closure: Microdeformations of Wounds and Cell Proliferation. *Plast. Reconstr. Surg.* 2004;114:1086-1096.

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